Carpel Tunnel Synovium: Does Clinical Abnormality Correspond with Histological Abnormality?

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Abstract

Background: Fibrous thickening of the flexor tendon synovium is a common finding during carpal tunnel decompression. This is generally accepted to indicate tenosynovitis. The aim of the study was to determine whether non-transparent, cloudy white thickening of the flexor tenosynovium corresponds to histological appearance of tenosynovitis.

Method: We retrospectively identified 49 wrists in 47 patients who underwent flexor tenosynovectomy during treatment for carpal tunnel decompression. The indication for synovectomy was clinical abnormality of the tenosynovium intra-operatively. Histological reports were retrospectively reviewed. A post-operative functional outcome score was compared between patients who underwent decompression alone and those who underwent decompression and synovectomy. Complication rates between the two groups were compared.

Results: Of the 49 slides analysed, inflammation was present in 10.1% (5 slides) only. Oedema was present in 51% (25 Slides). The mean functional outcome score for group 1 was 10 and 11.7 for group 2. This was statistically insignificant $p = 0.065$). The complication rates between the two groups were equal.

Conclusion: We conclude that clinical abnormality is a poor predictor of histological appearance of tenosynovitis.

Keywords: Carpal tunnel decompression; Flexor tendon synovium; Carpal tunnel synovium

Introduction

Compression of the median nerve in the carpal tunnel is the most common entrapment neuropathy. The multiple causes of carpal tunnel syndrome include anatomical abnormalities, inflammatory disorders, metabolic disease, fluid imbalances and trauma. All of these causes lead directly or indirectly to increase in volume of contents the carpal tunnel.

It is firmly established in literature that carpal tunnel syndrome is due to increase in carpal tunnel content’s volume which causes compression of the median nerve [1]. Fibrotic thickening of the flexor tendon synovium is a common finding during carpal tunnel decompression and is believed by some to be due tenosynovitis which could be chronic [2] or non-specific [3-8]. The commonly encountered pathological findings of the tenosynovium include fibrotic thickening of the sheath, intimal hyperplasia, vascular proliferation and thrombosis [9].

Lluch suggest that the thickening is a consequence rather than a cause of carpal tunnel syndrome [3]. Routine tenosynovectomy during carpal tunnel decompression has been at whether thickened appearance of the flexor tendon synovium is a positive indicator of histological recommended by some authors while others have not [10]. To knowledge there has been no study looking abnormality. The main aim of this study was to determine whether clinical impression of abnormality corresponded to histological abnormality defined as tenosynovitis. Functional outcome and complication rates were compared between a tenosynovectomy group and a similarly matched group of patients who underwent open carpal tunnel decompression without tenosynovectomy.

Method and Patient Demographics

Institutional review board was sought and obtained. 121 (in 108 wrists) open carpal tunnel decompressions where carried out over a twelve calendar month (2008). 49 wrists (in 47 patients) were retrospectively identified that underwent open carpal tunnel with partial flexor tenosynovectomy within the carpal tunnel wall. The indication for synovectomy was clinical abnormality defined as cloudy white appearance and non transparent thickening of the flexor tendon sheath.

The study group consisted of 14 men and 33 women with a mean age of 56.7 years. The syndrome was bilateral in 37 patients (79%). Of the 32 patients that worked, 7 did manual labour and 29 did non-manual labour. The average duration of symptoms before surgical decompression was 32 months. The most common pre-operative symptoms included parasthesias (73%), pain (51%) and weakness (10%).

The diagnosis of carpal tunnel decompression was made through a combination of history (paraesthesia in median nerve distribution in the hand, nocturnal hand pain), examination and electromyogram (EMG) studies (done in 42 out of 49 wrists).

In the EMG studies the motor latency was graded as follows, less than three milli-seconds was considered normal while three to four milli-seconds was considered mild, four to six point five milli-seconds was considered normal while three to four milli-seconds was considered mild, four to six point five milli-seconds was considered mild.
was deemed moderate and greater than six point five milli-seconds was considered severe. The 42 wrists that underwent EMG studies had positive (results 4 mild, 22 moderate and 16 severe). The remaining seven wrists that did not undergo EMG studies had previous history of carpal tunnel decompression in the contra-lateral wrist with satisfactory symptomatic outcome following surgery.

All patients underwent open surgical decompression under general anaesthesia by a single surgeon (senior author). Using sterile technique, the affected limb was draped and tourniquet was applied. A longitudinal incision of about three cm was made over the carpal tunnel in line with the third web space, and the palmar aponeurosis and transverse carpal ligament were incised in layers. Flexor tendon synovectomy was only carried out in cases where the senior author deemed the synovium to have a cloudy white, non transparent thickening. This was done as partial tenosynovectomy of the common flexor sheath within the carpal tunnel over the flexor digitorum superficialis tendons. Following the decompression and synovectomy, the tourniquet was released and haemostasis achieved with bipolar diathermy. The wound was closed with interrupted 4/0 nylon sutures and a volar splint applied. The splint was removed at one week post-operative and sutures were removed at week two.

The tenosynovia were sent to the Pathology Department for immediate fixation in formalin. The specimens were embedded in paraffin and stained with haematoxylin and eosin. The histological sections were analyzed for the following: inflammation, edema, vascular sclerosis, fibrosis and synovial hyperplasia. Inflammation of the synovial tissue was described as having either a perivascular or diffuse pattern and the presence of active inflammation was also recorded. Edema was classified as either absent, mild, moderate or severe. Vascular sclerosis, defined as hyaline degeneration of the blood vessels with hypertrophy of the tunica media and subintimal fibrosis was recorded as either present or absent. Fibrosis of the synovial tissue was recorded as either an absence of fibrosis or diffuse pattern. Synovial hyperplasia, described as hyperplasia and thickening of the layer of cells lining synovial tissue, was recorded as present or absent. These histological findings have proven to be the most consistent in previous studies on synovial tissue [1,2]. The Levine validated functional status outcome score, as described by Levine [11] was obtained post-operatively for the study group and the control group, matched for sex, age and time of surgery. Post operative complications were recorded for each group.

### Statistical methods

Between groups comparison were made using the Mann-Whitney U test. A p value of less than 0.05 was considered to be significant.

### Results

Inflammation was present in 10.2% of cases. A perivascular inflammatory infiltrate consisting of lymphocytes and plasma cells was the most common pattern seen (Figure 1), with a diffuse inflammatory infiltrate present in one case. One of these specimens also showed evidence of acute active inflammation with the presence of polymorphonucleocytes.

Edema was present in 51% of the patient specimens. The degree of edema was mild in 72% of cases, moderate in 28%.

Vascular sclerosis of small to medium sized vessels within the synovium proved to be the most common finding, affecting 77.5% of the sections.

The most frequent observations in the patient specimens were fibrosis and synovial hyperplasia. Fibrosis was observed in 4% of the specimens. Hyperplasia of the synovium was also present in only 2% of the patient specimens, and was associated with inflammation. Congo red stain proved positive for amyloid in 6.1% of cases.

At the minimal follow up of one year one patient in the study group had died of an unrelated cause to surgery (Leukaemia). An additional four patients were lost to follow-up, making 42 patients available for the post-operative functional outcome score. In the matched control, there were no deaths but eight patients were lost to follow-up making 33 available for the post-operative functional outcome score. There were 14 complications observed in both the control and study group (Table 1). All cases of infection were superficial and treated successfully with oral antibiotics. Scar sensitivity featured prominently in both groups and resolved with physiotherapy within a month.

The average time of operation, judged by the tourniquet time, was similar between the study group and control group 6.4 minutes and 5.7 minutes respectively. The mean functional outcome score for the tenosynovectomy group was 10, it was and 11.7 for the control group. Comparative analysis showed this to be insignificant p 0.065).

### Discussion

Increase in volume of the contents of the carpal tunnel is generally accepted to be a cause of carpal tunnel syndrome. The thickening of the synovium is a common finding during carpal tunnel decompression [2]. Lluch demonstrated in his experiments with rabbits that thickening
of the synovium was a consequence of increased intra-carpal tunnel pressures rather than a cause of the syndrome [3]. The role of tenosynovectomy in carpal tunnel decompression is debated and while thickening of the synovium is a common finding, many authors do not recommend routine synovectomy as part of open decompression [12]. Some authors recommend tenosynovectomy in revision operations [13] or in patients involved with heavy manual or highly repetitive work [14].

In our practice, tenosynovectomy is not done routinely but is considered in cases where there is clinical suspicion of tenosynovitis, although tenosynovitis has been shown to be a rare finding in histological analysis of the flexor tendon synovium during open carpal tunnel decompression [2,3,7]. The main aim of this study was to determine whether the presence of thick, cloudy white appearance of the flexor tendon synovium would be a higher predictive indicator of histology appearance of tenosynovitis. Of the 49 slides examined only five (10.1%) showed signs of inflammation. A large portion of the slides showed non-specific changes including vascular proliferation (77.1%) and edema (51%). These findings, especially that of edema, has been previously reported in literature [15]. We believe these non-specific features may occur as a consequence of increase in volume causing local ischemia with resultant synovial edema from increased vascular permeability which is supported by the high rate of vascular proliferation in this study.

The functional outcome score described by Levine et al. is particularly useful as it has been validated for carpal tunnel decompression. We found the mean score between the tenosynovectomy group (study group) and the control group that underwent open decompression without tenosynovectomy was 10 and 11.7 respectively. Between Tests comparative analysis showed this to be statistically insignificant (p = 0.065). The incidence of complications between the two groups was 33% in the study group and 42% in the control group. Scar sensitivity was the most common finding in both groups. This was treated successfully with desensitization techniques which included regular massage of the scar with emollients. These findings suggest that synovectomy for clinical appearance of abnormality is a poor indicator of histological presence of tenosynovitis. Our study is limited by being retrospective; as such no pre-operative Levine Score was available. A prospective study with additional sampling from the non-involved wrist may help determine if the finding of 10.1% inflammation is truly evidence of pathology or a natural finding. We conclude that clinical appearance of abnormality is a poor indicator of tenosynovitis.

References